We claim:

1. A method for preventing degradation in functional performance of motor or sensory nerves in an animal comprising administering to the animal a therapeutic amount of a *hedgehog* or *ptc* therapeutic.

2. A method for preventing dysfunction of motor or sensory nerve cells comprising contacting the cells with an effective amount of a *hedgehog* or *ptc* therapeutic.

3. A method for treating or preventing peripheral neuroathy comprising administering to an animal a protective amount of a *hedgehog* or *ptc* therapeutic.

4. A method for protecting peripheral nerve cells under conditions which otherwise result in peripheral neuropathy, compriseing administering to a patient in need thereof a therapeutically effective amount of a *hedgehog* or *ptc* therapeutic.

5. A method for the treating or preventing diabetic neuropathy comprising administering to a patient in need thereof a therapeutically effective amount of a *hedgehog* or *ptc* therapeutic.

6. A method for the treating or preventing virally-induced peripheral neuropathy comprising administering to a patient in need thereof a therapeutically effective amount of a *hedgehog* or *ptc* therapeutic.

7. The method of any of claims 1-6, wherein the *hedgehog* therapeutic is a polypeptide which includes a hedgehog amino acid sequence which is identical or homologous to an amino acid sequence of any one of SEQ ID Nos. 10-18.

8. The method of claim 7, wherein the hedgehog amino acid sequence is sufficient for specific binding of the polypeptide to a *patched* protein.

9. The method of claim 7, wherein the hedgehog amino acid sequence is at least 80 percent identical to an amino acid sequence of any one of SEQ ID Nos. 10-18.

10. The method of claim 7, wherein the hedgehog amino acid sequence is encodable by a nucleic acid which hybridizes under stringent conditions to any one of SEQ ID Nos. 1-9.

11. The method of claim 7, wherein the hedgehog amino acid sequence is of a vertebrate hedgehog protein.

12. The method of claim 11, wherein the vertebrate hedgehog protein is *Dhh*.

13. The method of claim 7, wherein the polypeptide includes at least a 50 amino acid extracellular portion of a vertebrate hedgehog protein.

14. The method of claim 7, wherein the polypeptide includes at least a 150 amino acid extracellular portion of a vertebrate hedgehog protein.

15. The method of claim 7, wherein the polypeptide includes at least an extracellular portion of a vertebrate hedgehog protein corresponding to residues 24-194 of SEQ ID No:15.

16. The method of claim 7, wherein the hedgehog polypeptide is modified with one or more lipophilic moieties.

17. The method of claim 16, wherein the hedgehog polypeptide is modified with one or more sterol moieties.

18. The method of claim 17, wherein the sterol moiety is cholesterol.

19. The method of claim 16, wherein the hedgehog polypeptide is modified with one or more fatty acid moieties.

20. The method of claim 19, wherein each fatty acid moiety is independently selected from the group consisting of myristoyl, palmitoyl, stearoyl, and arachidoyl.

21. The method of claim 16, wherein the hedgehog polypeptide is modified with one or more aromatic hydrocarbons.

22. The method of claim 21, wherein each aromatic hydrocarbon is ondependently selected from the group consisting of benzene, perylene, phenanthrene, anthracene, naphthalene, pyrene, chrysene, and naphthacene.

23. The method of claim 16, wherein the hedgehog polypeptide is modified one or more times with a C7 - C30 alkyl or cycloalkyl.

24. The method of of any of claims 1-6, wherein the *ptc* therapeutic is a small organic molecule.

25. The method of claim 24, wherein the binding of the ptc therapeutic to *patched* results in upregulation of patched and/or gli expression.

26. The method of any of claims 1-6, wherein the *ptc* therapeutic binds to *patched* and mimics *hedgehog*-mediated *patched* signal transduction.

27. The method of claim 26, wherein the *ptc* therapeutic is a small organic molecule.

28. The method of claim 26, wherein the binding of the ptc therapeutic to *patched* results in upregulation of patched and/or gli expression.

29. The method of any of claims 1-6, wherein the *ptc* therapeutic is a small organic molecule which interacts with neuronal cells to mimic *hedgehog*-mediated *patched* signal transduction.

30. The method of any of claims 1-6, wherein the *ptc* therapeutic mimics *hedgehog*-mediated *patched* signal transduction by altering the localization, protein-protein binding and/or enzymatic activity of an intracellular protein involved in a *patched* signal pathway.

31. The method of any of claims 1-6, wherein the *ptc* therapeutic alters the level of expression of a *hedgehog* protein, a patched protein or a protein involved in the intracellular signal transduction pathway of *patched*.

32. The method of claim 31, wherein the *ptc* therapeutic is an antisense construct which inhibits the expression of a protein which is involved in the signal transduction pathway of *patched* and the expression of which antagonizes *hedgehog*-mediated signals.

33. The method of claim 32, wherein the antisense construct is an oligonucleotide of about 20-30 nucleotides in length and having a GC content of at least 50 percent.

34. The method of claim 33, wherein the antisense oligonucleotide is selected from the group consisting of: 5'-GTCCTGGCGCCGCCGCCGCCGTCGCC;

5'-TTCCGATGACCGGCCTTTCGCGGTGA; and

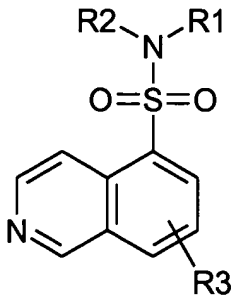
5'-GTGCACGGAAAGGTGCAGGCCACACT

35. The method of claims 31, wherein the *ptc* therapeutic is a small organic molecule which binds to *patched* and regulates *patched*-dependent gene expression.

36. The method of claim 35, wherein the *ptc* therapeutic is an inhibitor of protein kinase A.

37. The method of claim 36, wherein the PKA inhibitor is a 5-isoquinolinesulfonamide

38. The method of claim 37, wherein the PKA inhibitor is represented in the general formula:



wherein,

R_1 and R_2 each can independently represent hydrogen, and as valence and stability permit a lower alkyl, a lower alkenyl, a lower alkynyl, a carbonyl (such as a carboxyl, an ester, a formate, or a ketone), a thiocarbonyl (such as a thioester, a thioacetate, or a thioformate), an amino, an acylamino, an amido, a cyano, a nitro, an azido, a sulfate, a sulfonate, a sulfonamido, $-(CH_2)_m-R_8$, $-(CH_2)_m-OH$, $-(CH_2)_m-O$-lower alkyl, $-(CH_2)_m-O$-lower alkenyl, $-(CH_2)_n-O-(CH_2)_m-R_8$, $-(CH_2)_m-SH$, $-(CH_2)_m-S$-lower alkyl, $-(CH_2)_m-S$-lower alkenyl, $-(CH_2)_n-S-(CH_2)_m-R_8$, or

R_1 and R_2 taken together with N form a heterocycle (substituted or unsubstituted);

R_3 is absent or represents one or more substitutions to the isoquinoline ring such as a lower alkyl, a lower alkenyl, a lower alkynyl, a carbonyl (such as a carboxyl, an ester, a formate, or a ketone), a thiocarbonyl (such as a thioester, a thioacetate, or a thioformate), an amino, an acylamino, an amido, a cyano, a nitro, an azido, a sulfate, a sulfonate, a sulfonamido, $-(CH_2)_m-R_8$, $-(CH_2)_m-OH$, $-(CH_2)_m-O$-lower alkyl, $-(CH_2)_m-O$-lower alkenyl, $-(CH_2)_n-O-(CH_2)_m-R_8$, $-(CH_2)_m-SH$, $-(CH_2)_m-S$-lower alkyl, $-(CH_2)_m-S$-lower alkenyl, $-(CH_2)_n-S-(CH_2)_m-R_8$;

R_8 represents a substituted or unsubstituted aryl, aralkyl, cycloalkyl, cycloalkenyl, or heterocycle; and

n and m are independently for each occurrence zero or an integer in the range of 1 to 6.

39. The method of claim 36, wherein the PKA inhibitor is cyclic AMP analog.

40. The method of claim 36, wherein the PKA inhibitor is selected from the group consisting of N-[2-((p-bromocinnamyl)amino)ethyl]-5-isoquinolinesulfonamide, 1-(5-isoquinoline-sulfonyl)-2-methylpiperazine, KT5720, 8-bromo-cAMP, dibutyryl-cAMP and PKA Heat Stable Inhibitor isoform α.

41. The method of any of claims 4-6, wherein patient is being treated prophylactically.

42. A therapeutic preparation of a small molecule antagonist of *patched*, which *patched* antagonist is provided in a pharmaceutically acceptable carrier and in an amount sufficient to treat a peripheral neuropathy.

43. A method for protecting peripheral nerve cells under conditions which otherwise result in peripheral neuropathy, comprising administering to a patient a gene activation construct which recombines with a genomic *hedgehog* gene of the patient to provide a heterologous transcriptional regulatory sequence operatively linked to a coding sequence of the *hedgehog* gene.

44. The method of claim 4, 5, 6 or 43, which method is part of a protocol for the treatment of an acquired neuropathy.

Sub D13

45. The method of claim 44, wherein the neuropathy is due to viral infection, diabetes or inflamation.

46. The method of claim 44, wherein the neuropathy is due to contact with a toxic agent.

Sub D14 D10

47. The method of claim 44, wherein the neuropathy is selected from the group consisting of diabetic neuropathy; immune-mediated neuropathy, chronic inflammatory demyelinating polyneuropathy (CIDP), chronic polyneuropathy with antibodies to peripheral nerves, neuropathies associated with vasculitis or inflammation of the blood vessels in peripheral nerve, brachial or lumbosacral plexitis, and neuropathies associated with monoclonal gammopathies; neuropathies associated with tumors or neoplasms such as sensory neuropathy associated with lung cancer, neuropathy associated with multiple myeloma, neuropathy associated with waldenstrom's macroglobulemia, chronic lymphocytic leukemia, or B-cell lymphoma; neuropathy associated with amyloidosis; neuropathies caused by infections; neuropathies caused by nutritional imbalance; neuropathy in kidney disease; hypothyroid neuropathy; neuropathy caused by alcohol and toxins; neuropathies caused by drugs; neuropathy resulting from local irradiation; neuropathies caused by trauma or compression; and idiopathic neuropathies

48. The method of claim 4, 5, 6 or 43, which method is part of a protocol for the treatment of a hereditary neuropathy.

49. The method of claim 48, whererin the neuropathy is selected from the group consisting of Charcot-Marie Tooth Disease (CMT); Familial Amyloidotic Neuropathy and Hereditary Porphyria.

50. The method of claim 4, 5, 6 or 43, which method is part of a protocol for slowing neurodegenerative events associated with age-related neuropathology.

Sub D15

51. The method of claim 7, wherein the hedgehog polypeptide is a fusion protein.

add D16

Add E9